AN EXTENSION OF THE STUDY OF SERUM LIPOPROTEIN PROFILE DURING THE PROCESS OF LABOUR SPONTANEOUS/INDUCED WITH SPECIAL REFERENCE TO THE METHOD OF TERMINATION OF PREGNANCY

THESIS

For

MASTER OF SURGERY

(Obstetrics & Gynaecology)

D11447

Approved.

BUNDELKHAND UNIVERSITY
JHANSI (U. P.)

1994 **JUHI ARORA**

NO

SUCCESS

IS

APART FROM MY

MOTHER

TO

WHOM

I OWE EVERYTHING

AND

TO

HER I DEDICATE

THIS WORK

C E R T I F I C A T E

This is to certify that the work entitled "AN EXTENSION OF THE STUDY OF SERUM LIPOPROTEIN PROFILE DURING THE PROCESS OF LABOUR SPONTANEOUS/INDUCED WITH SPECIAL REFERENCE TO THE METHOD OF TERMINATION OF PREGNANCY", which is being submitted as a thesis for M.S.(Obstetrics and Gynaecology) by DR. JUHI ARORA, has been carried out under my direct supervision and guidance in the department of Obstetrics and Gynaecology M.L.B. Medical College, Jhansi.

She has put in the necessary stay in the department as per university regulations.

Dated: 7.1.94

Sunita Arora

(Sunita Arora)
Associate Professor & Head,
Department of Obst. &
Gynaecology,
M.L.B. Medical College,
Jhansi.

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Sunita Arora

Dated: 7.1.94

(Sunita Arora)
M.S.,
Associate Professor,
Department of Obstetrics & Gynaecology,
M.L.B. Medical College,
JHANSI.

(GUIDE)

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Dated: 7.1.94

(R. C. Arora)
M.D., D.Sc.,
Professor and Head,
Department of Medicine,
M.L.B. Medical College,
Jhansi.

(CO-GUIDE)

2

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Dated: 1.1.94

Agarwal

(Navnit Agarwal)
M.D.,
Assistant Professor,
Department of Medicine,
M.L.B. Medical College,
JHANSI.

(CO-GUIDE)

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Dated: 4·1·94

(Usha Agarwal)
M.S.,
Associate Professor,
Department of Obstetrics &
Gynaecology,
M.L.B. Medical College,
JHANSI.

(CO-GUIDE)

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Dated:

Juhi

(Juhi Arora)

C O N T E N T S

INTRODUCTION

INTRODUCTION

Human pregnancy is accompanied by pronounced alterations in carbohydrate and lipid metabolism. The content of triglyceride, cholesterol and phospholipids in all three main lipoprotein fractions increases and elevation also occurs in the lipoprotein apoprotein concentrations. Thus maternal hypertriglyceridemia is one of the most striking and consistent change occurring at late gestation in both human and animals. The physiological significance is not yet completely understood.

In normal pregnancy serum total cholesterol and triglyceride levels steadily increase starting from second trimester upto term and decreases after delivery (Oliver Boyd, 1933; Duckman and Wegner, 1934; Schwarz et al, 1940; Watson, 1957). These increased levels of serum total cholesterol which increases with advancing pregnancy attain a maximum peak just prior to the onset of labour and then abruptly falls with the expulsion of placenta and fetus, but not reaching the pregestational levels. In the postpartum phase the serum total cholesterol level falls gradually.

These changes in lipoprotein profile during pregnancy mirror those seen with oral contraceptive, users and during treatment with sex hormones, strongly suggesting and supporting a hormonal basis for the hyper-

lipidemia of pregnancy. Thus the endocrine changes during gestation have been proved to be the causative factor altering the lipid metabolism.

The fetaplacental unit contributes to the rise in serum levels of oestrogen and progesterone in pregnant women, which may be to a certain extent account for the variation of lipid parameters.

The hypertriglyceridemia that occurs during pregnancy falls postpartum. Although studies are available for the antepartum and postpartum phase not much has been done during the intrapartum phase. Study is lacking regarding how the process of labour alters these lipoprotein levels.

Moreover, much of the data available is for the white females which cannot serve as a standard parameter for Indian females in view of the fact that lipoprotein levels are altered by diet, socioeconomic status, familial pattern, smoking, alcohol ingestion and lactation.

This work has been directed to study the alterations in the lipoprotein profile during the actual process of labour - through stage first, second and third. To observe the trend of the falling lipoprotein levels during labour and to know where exactly the fall occurs and also how these changes are altered when the process of labour is curtailed by caeserian section.

With the ongoing work this study has been extended to include the changes in the lipid paramters during the first twenty four hours postpartum as well. With all these informations the present study is being made with the following objectives.

1. To study the various changes in lipoprotein profile during antepartum, intrapartum and postpartum phases of pregnancy and correlate it with variables such as socio-economic status, diet, parity, disease status, drug intake.

2. To compare the observed lipoprotein changes in spontaneous vaginal delivery to :

   a. Artificially induced delivery by -

      i) Artificial rupture of membranes (ARM).

      ii) Oxytocin infusion.

      iii) Prostaglandins.

   b. Caeserean delivery

      i) Elective .

      ii) Emergency.

3. To study if any relation of lipoprotein levels exists or altered by -

   - Duration of gestation.
   - Duration of labour.
   - Drugs administered during labour.

5. To study the changes in the lipoprotein profile during the first 24 hours of postpartum.
6. To study the variations in the changes in lipoprotein profile during the postpartum phase in vaginal delivery

REVIEW OF LITERATURE

R E V I E W   O F   L I T E R A T U R E

Pregnancy is attended by extensive hormonal readjustments on the part of the mother. Almost every endocrine tissue participates in the adaptive changes, that maintain the metabolic state of the female during normal pregnancy.

The hyperlipidemia of late pregnancy was first described in 1845 (Bacqueral and Rodier). Virchow (1847) showed that the milky appearance of the sera of the pregnant women was due to the presence of fat. The first chemical study was undertaken in 1911 when Chanfford and associates demonstrated an increase of blood cholesterol during normal pregnancy.In the same year, Neumann and Hermann studied the lipid particles in whole blood and reported increase in cholesterol during pregnancy. They concluded that during the first 6-7 months, the serum cholesterol might be increased and that during the last 2 months the increase in serum cholesterol was a rule.

Plase and Tompkins (1923) have also given figures for the blood and lipids particularly cholesterol during pregnancy. These figures indicate a gradual rise from fourth month to term.

Tyler and Underhill (1925) determined whole blood cholesterol in normal and in pregnant women. They studied gravid women in each month of pregnancy, beginning from

the third and stated that cholesterol and ester cholesterol increase gradually until term when it is roughly one third higher than at three months.

Gardner and Gainsborough (1929) studied the ratio of cholesterol to cholesterol esters and reported an increase in cholesterol with a decrease in cholesterol ester till the 30th week of pregnancy. In their series reversal of the relationship occurs so that at parturition approximately a normal relationship exists again.

Kaufmann and Mihlbock (1933) did not notice these fluctuations but they reported little variations from the second months of gestation to term.

Diecmann and Weiguer (1934) found that the total cholesterol increases to 23% above the first trimester levels and which decreased to 27% at the eighth postpartum week from the values noted at term.

Oliver and Boyd (1935) after careful study of 12 normal primigravida stated that there occurs a highly significant rise in plasma ester and total cholesterol between 31st and 33rd weeks of pregnancy. By the 20th postpartum week these values have decreased considerably but were all higher than the levels at 12th week of pregnancy.

De Alvarez et. al (1953) have however found values much higher than Diecmann and Weigner. They have demonstrated a 54% increase in third trimester values of serum

cholesterol above the first trimester and a 23% decrease in the values 6-7 weeks postpartum as compared to third trimester values.

Dannenburg et al (1962) made daily serial estimations of the plasma esterified fatty acids on puerperal patients following delivery and through fifth postpartum day. They found that the mean puerperal values for total carboxyl esters were higher than the nonpregnant class, and this difference was statistically significant. All fractions of the esterified fatty acids except cholesterol ester showed a slight increase within 24 hours of delivery and then subsequently declined. These changes were greatest in the triglycerides and least in esterified cholesterol. This agrees with the observations of Boyd (1935) and Schwarz (1940) but contrary to other reports. Peters (1951), Wastson (1957), De Alvarez (1959) showed a drop in plasma lipids following delivery.

Mullick and Bagga (1964) made a two phase study to define more precisely the changes in total lipids and their fractions during different periods of gestation and also to analyse the factors which bring about these changes. They concluded that all lipid fractions show a gradual and persistent rise throughout pregnancy. The beta lipoprotein and alpha lipoprotein ratio increases as pregnancy advances.

Knottinan and Pyorala (1964) studied serum lipids in late pregnancy, at delivery and during early puerperium in mothers with normal pregnancy and those with pre-

eclampsia. Levels which were numerically higher but not statistically significant were seen in preeclamptic except at delivery when a statistically higher levels were observed in the latter group.

Bhattacharya (1969) concluded after their extensive study over normal and abnormal pregnancy that although cholesterol levels were slightly higher in toxemia group. The cholesterol metabolism seemed to be similar in normal and toxemia of pregnancy.

Potnis, Gupte and Purandare (1977) studied the antepartum and postpartum cholesterol and lipoprotein level changes, determined its significance and correlation to the hormonal changes at parturition. They concluded that the endocrine changes during gestation may be the cause for the alterations in lipid metabolism with the extirpation of placenta and hence the removal of source of hormones being responsible for the decline.

Ronald K. Kalkhoff (1978) studied carbohydrate and lipid metabolism during normal pregnancy and their relationship to gestational hormone action and stated that the hypertriglyceridemia of late pregnancy is mainly due to increase in VLDL concentration. Hypertriglyceridemia is also due to increase in HDL and LDL. They also stated that oestrogen is the principal hormonal factor responsible for increased synthesis and release of endogenous triglycerides. Enhanced triglycerides removed during mid gestation may be due to the rising level of progesterone.

Potter and Nestel (1979) studied lipoprotein profile during pregnancy and puerperium in a group of 43 women. The plasma cholesterol concentration rose on the average by about 50%, major increase occurring in second trimester. The plasma triglyceride concentration rose three fold reaching its peak during the third trimester.

Glueck et al (1980) have tried to determine the inter-relationship between pregnancy, hypertriglyceridemia and pancreatitis. They concluded that routine quantitation of plasma cholesterol and triglyceride levels should be done during early pregnancy so as to identify the women with severe familial hypertriglyceridemia prior to the superimposition of the physiologic hyperlipidemia of pregnancy so as to overt the sometime catastrophic sequelae.

Darmady and Postle (1982) monitored the serum lipid concentration in a group of normal women from before conception throughout gestation and until at least 40 weeks after delivery. The effect of lactation was also examined. The primary change in lipoprotein metabolism during pregnancy appears to be concerned with VLDL which are elevated, the rate of secretion depending upon the lipoprotein lipase activity. After delivery the elevated serum triglyceride concentration decreases rapidly and the significantly greater utilization of serum triglyceride in lactating women could be caused by the tissue specific direction of VLDL towards the mammary gland for milk synthesis.

Knopp et al (1982) have tried to define a population based lipoprotein lipid reference value for pregnant women compared to nonpregnant women classified by sex hormone usage. The percentile distributions of lipoprotein levels presented have clinical and research application. The ninety fifth percentile value (387 mg/dl) and the ninety ninth percentile value (500 mg/dl) for whole plasma triglycerides indicate the magnitude of the triglyceride elevation in pregnancy. These values can also be used to detect underlying hypertriglyceridemia and the susceptibility of the patients to develop hyperlipemia later in life. The first and fifth percentile levels of HDL-c of 35 and 42 mg/dl represent abnormally low concentration in pregnancy typically associated with endogenous hypertriglyceridemia and accelerated heart disease risk.

Fahraeus Lars et al (1985) studied the levels of plasma lipoprotein fractions in 19 healthy women at exact gestational ages. The HDL levels elevated in the 14th week and showed a maximum rise of 41% in the 28th week of pregnancy. The LDL decreased in early pregnancy but then increased continuously. The VLDL concentration showed a continuous increase from week 14 to week 36. During lactation eight weeks after delivery the LDL concentration remained elevated whereas the other lipoproteins had returned to prepregnancy levels.

Herrera and Armda (1988) have tried to study the basic mechanism involved in maternal hypertriglyceridemia

in late normal pregnancy and its physiologic significance reviewed as a model of the effects of sex steroids in lipoprotein metabolism. They concluded that changes in magnitude and even the direction of lipoprotein lipase activity in different tissues during gestation actively contribute not only to the development of hypertriglyceridemia but also to the metabolic fate of circulating triglycerides. Any change in these dynamic and intricate metabolic adaptations seen in the mother may directly modify her lipoprotein profile. Under pathologic conditions the alterations may be permanently maintained thereby increasing the risk for the development of cardiovascular disease.

Valimaki et al (1990) have studied the serum lipids and lipoproteins in alcoholic women during pregnancy. They concluded that alcohol abuse clearly reduced the normal increase in total and LDL cholesterol during the 24 to 40th weeks. LDL changes were most pronounced in alcoholic women who later produced FAS infants. HDL-3 was raised and normal increase in VLDL was accentuated in these women.

Arora and Neeta (1993) studied the changes in lipoprotein profile in normal pregnancy and artificial termination of pregnancy (Elective/Emergency LSCS). They observed a rising trend in lipoprotein profile with a peak during labour followed by a fall in the postpartum period, both in normal as well as artificial termination of pregnancy.

The umbilical cord blood values of lipoproteins specially of serum total cholesterol and serum triglyceride were higher in patients of emergency caeserian section. They concluded this variation to the vigorous uterine contractions pushing large amount of lipoproteins from maternal blood to umbilical cord. Statistically significant low levels were encountered in patients in whom pregnancy had been terminated by elective LSCS where these contractions are absent.

LIPIDS

The lipids are a heterogenous group of compounds related actually or potentially to the fatty acids. They are an important dietary constituent, not only because of their energy value but also because of the fat soluble vitamins and the essential fatty acids contained in the fat of natural foods. In the body, fat serves as an efficient source of energy. Combination of fat and protein(lipoprotein) are important cellular constituent and serve as the means of transporting lipids in the blood.

CLASSIFICATION

The following classification has been suggested by Bloor.

A. SIMPLE LIPIDS : Esters of fatty acids with various alcohols.- (i) Fats, (ii) Waxes.

B. <u>COMPOUND LIPIDS</u> : Esters of fatty acids containing groups in addition to an alcohol and a fatty acid.

(i) Phospholipids, (ii) Glycolipids

(iii) Others - These include sulfolipids, and amino lipids. Lipoproteins are also included in this group.

C. <u>DERIVED/PRECURSOR LIPIDS</u> : These include substances derived from the above groups by hydrolysis. They include fatty acids, glycerol, steroids, ketone bodies, lipid soluble vitamins and hormones.

<u>CHOLESTEROL</u>

Synthesized in all nucleated cells, it is a sterol containing hydrogenated phenanthrene ring. 70-80% of serum total cholesterol is in ester form and 20-30% forms free cholesterol. Thus one third of the total daily synthesis - about 800 mg formed in liver is transported as beta lipoproteins. The serum levels range from 130-260 mg%.

<u>TRIGLYCERIDES</u>

Also called neutral fats are esters of the alcohol glycerol and fatty acids. Triglycerides form the main bulk of dietary lipids. About 1-2 gm/kg body weight of triglycerides are ingested daily (Heury, 1977). During metabolism they are broken into di and mono-glycerides and fatty acids. After absorption the fatty acids are again converted into triglycerides. The normal values for adult range from 80-240 mg%.

PHOSPHOLIPIDS

The adult blood levels range between 150-250 mg%. They include mainly phosphatidyl choline, sphingomyelin, phosphatidyl ethanolamine and lipophosphatidyl-choline. The proportions of these vary in the different lipoproteins.

FATTY ACIDS

The non esterified forms constitute the active fraction and called as free fatty acids (FFA). The blood levels range between 8-30 mg%.

LIPOPROTEINS

The lipids are present in the plasma mainly in the form of lipoproteins which serve the two well established functions: the transport of triacylglycerol and the transport of cholesterol and its esters. The free fatty acids are bound to albumin. Lipoproteins constitute aggregation of lipids with other proteins. The protein part of lipoprotein is called Apoproteins. The lipid content decreases the density of lipoproteins. The differences in the content of lipids and proteins among the several lipoproteins give them different densities and permit their further separation in centrifuge.

Five major groups of lipoproteins have been identified . These include :-

a. Chylomicrons.

b. Very low density lipoproteins (VLDL) or prebeta lipoproteins.

c. Low density lipoproteins (LDL) or beta lipoprotein.

d. High density lipoproteins (HDL) or alpha lipoprotein.

e. Free fatty acids.

a. CHYLOMICRONS

These are formed only by the lymphatic system draining the intestine. The levels fluctuates with the load of triacylglycerol absorbed. They are cleared rapidly from the circulation within one hour.

b. VLDL OR PREBETA LIPOPROTEINS

These consists mainly of glycerides that are endogenous i.e. newly synthesised or derived from body stores rather than diet. It is formed by the hepatic parenchymal cells. The formation is quantitatively less but is more constant and occurs even in the fasting state. It serves as a vehicle of transport of triacylglycerol from the liver to the extrahepatic tissues.

During metabolism virtually all of the VLDL is converted to LDL. The levels of VLDL between 20-29 years, is about 25%mg% and between 30-39 years - 35 mg%. Half life of VLDL is about 2 hours.

c. LOW DENSITY LIPOPROTEINS(LDL) - BETA LIPOPROTEINS

It is mainly derived from breakdown of VLDL in circulation. Depending upon the densities they are further divided into LDL_1 and LDL_2 types. The overall cholesterol concentration in plasma is a reflection of the LDL concentration. LDL is relatively stable with a much longer half life of about 4 days. In man the receptor sites are

saturated with LDL which accumulates in plasma and is only slowly cleared. Levels range between (170-190 mg%) between 20-40 years.

d. HIGH DENSITY LIPOPROTEINS(HDL)- ALPHA LIPOPROTEIN

These are isolated between densities of 1.063 - 1.210 and contain about 50% protein. They are much smaller than the other lipoproteins. HDL either scavenges cholesterol from peripheral tissues or it helps in destruction of IDL and LDL. HDL is secreted from liver. It contains only free cholesterol which is later esterified. It may subsequently pick up free cholesterol from cell membranes rather than cholesterol ester. HDL has a negative co-relation to coronary artery disease. The blood levels of HDL in females between 20-29 years is 75 mg% and that between 30-39 years - 80 mg%.

Comparison of the lipoprotein in plasma of man (Adapted from Olson and Vester, 1960)

Fraction	Source	Composition (%)						
		Protein	Total lipids	Triglycerides	Phospholipids	Cholesteryl ester	Cholesteral free	Free fatty acids
1. Chylomicrons	Intestine	1-2	98-99	88	8	3	1	-
2. Very low density lipoproteins (VLDL)	Liver and intestine	7-10	90-93	56	20	15	8	1
3. Low density lipoproteins LDL or IDL	VLDL chylomicrons	11	89	29	26	34	9	1
LDL_2		21	79	13	28	48	10	1
4. High density lipoproteins HDL_1	Liver, ? Intestine							
HDL_2		33	67	16	43	31	10	-
HDL_3		57	43	13	46	29	6	6

HORMONAL CHANGES DURING PREGNANCY AND POSTPARTUM PERIOD AND THEIR EFFECT OVER LIPID METABOLISM

Pregnancy is accompanied by profound hormonal alterations which persist for the duration of pregnancy. It is well known that hormones influence the level of lipids. In the early weeks the corpus Luteum of pregnancy serves as a source of hormones. Soon the action is taken over by the placenta which serves as the main endocrine organ bringing about continued and higher production of steroid and other hormones. Besides, the anterior pituitary, adrenal cortex also have an important role to play in the extrapolation of hormones, which support pregnancy.

A. PLACENTAL HORMONES

1. OESTROGENS

Oestriol is the main pregnancy oestrogen accounting for 80-90% of the total oestrogen formed. The oestrogen levels during pregnancy increases progressively to reach a level of approximately 150 mg/ml at terin (Ronald K. Kalkhoff et al, 1978), a level almost 16 times higher than the values at 8 weeks (Desoye et al, 1986).

Oestrogen levels fall significantly within 3 days and reach a basal level by 7th postpartum day, rising again in nonlactating by the 14th day.

Estrogens cause an increase in HDL-c while LDL is decreased. The biosynthesis of VLDL is enhanced but the triglyceride lipase activity reduced.

2. PROGESTERONE

After the first trimester, the placenta becomes capable of producing sufficient progesterone to maintain gestation, the levels of which in maternal plasma increases progressively with gestation. Cholesterol derived from maternal blood is the main substrate for the trophoblastic synthesis of progesterone. The levels of the hormone secreted by the placenta approximates 250 mg/day, the levels at term being 7 times the value at 8 weeks.

Progesterone levels fall significantly within 3 days reaching basal levels by 7th day in lactating mothers.

The oestrogen to progesterone ratio is also increased from 0.08 in the first trimester to a value of 0.232 at about 35 weeks. Thereafter it decreased to 0.187 at 38 weeks. No association has been found between lipid and lipoprotein levels to the oestrogen to progesterone ratio, except that the fall of ratio was parallel to the fall in LDL levels immediately before term (Desoye et al, 1986).

Individually progesterone brings about a decrease in HDL cholesterol and an increase in LDL-c . It induces hepatic triglyceride lipase activity. Increased degradation of VLDL and/or IDL resulting in high plasma LDL-c levels.

3. HUMAN PLACENTAL LACTOGEN

A polypeptide hormone secreted by the placenta gradually increases and eventually reaches a maximum of

5-8 ugm/ml at term. Maternal concentrations promptly return to undetectable levels within 24 hours.

The levels of HPL clearly parallel the time course of the lipids changes during pregnancy. It has lipolytic activity thus releasing FFA probably by activation of hormone sensitive lipase. This may occur for foetal requirements in the second half of pregnancy during which the mass of maternal adipocytes are reduced and the foetus gains weight. The portion of free fatty acids not utilised by foetus is incorporated into STG and VLDL in maternal liver (Desoye et al, 1987).

4. HUMAN CHORIONIC GONADOTROPIN (HCG)

Concentration of HCG rises to peak values by 8-12 weeks of gestation. Thereafter there is a decrease in HCG levels to a plateau that is maintained throughout the remainder of pregnancy. It becomes undetectable in urine by 7-10 days postpartum. Free cholesterol was inversely related to HCG levels whereas triglyceride concentration resembled those to insulin.

B. ANTERIOR PITUITARY HORMONES

1. HUMAN GROWTH HORMONE (HGH)

Basal levels of growth hormone are low during early pregnancy and do not change remarkably with advancing gestation. Pituitary somatotrophic hormone or growth hormone raises the blood lipid levels. Slow rise in HGH occurs during postpartum phase.

2. PROLACTIN

Concentration of serum prolactin in pregnancy begin to increase approximately 30 days after the mid menstrual cycle peak of lutienising hormone. Rising prolactin levels continue to increase to reach peak levels at term. Serum prolactin declines rapidly after parturition, if the woman does not breast feed. However, prolactin levels increase sharply with breast feeding episodes, they then decrease to non pregnant values after several months of lactation.

C. THYROID HORMONES

1. THYROXIN

Like oestrogens it depresses the blood lipid levels. Patterson, Hund and Nicodens (1938) believed that hypercholesterolemia of pregnancy is due to subclinical hypothyroidism.

Lister (1955) and Russell (1956) found that protein bound iodine and serum precipitate iodine are elevated as early as second month of pregnancy. These levels have been found to reach as high as those seen in individual with overt hyperthyroidism.

Strisower (1958) found that the thyroid hormone depresses serum lipid partition but during pregnancy the tissue become more refractory to the effect of thyroxin.

D. ADRENAL HORMONES

GLUCOCORTICOIDS

Cortisol metabolism is significantly altered during pregnancy, the maternal plasma levels rising progressively throughout gestation. The plasma levels of transcortin also rises progressively to a peak in third trimester. The increased transcortin levels are due to increased oestrogen concentration. The maternal tissues are exposed to an average daily concentration of cortisol that is more than twice normal. Cortisone increases the cholesterol and its ester level and thus is achieved at the cost of neutral fats (Jailer et al, 1957).

E. PANCREATIC HORMONES

INSULIN

The basal levels of insulin tend to become progressively higher as term gestation is approached. Also a much greater amount of insulin is released in response to glucose stimulation. However, during pregnancy a state of insulin resistance exists. Insulin has a pronounced antilipolytic effect and antagonises the lipolytic effect of hormones mainly by inhibiting the hormone sensitive lipase in the adipose tissue. Thus it reduces the release not only of free fatty acids but of glycerol as well.

EFFECT OF DIET ON SERUM LIPOPROTEIN PROFILE

In 1929, Gardner and Gainsborough carried out complete studies on cholesterol metabolism and concluded that during periods of fasting, cholesterol content of plasma varies markedly in different healthy persons but is fairly constant in subject. A single meal does not cause any change but prolonged diets high or low in sterols will cause variation in cholesterol. The free cholesterol remains fairly constant but cholesterol esters shows greatest change.

According to Mullick and Bagga (1964), in healthy females, serum lipid levels and its fraction varies with the nutritional status, which is itself dependent upon the socio-economic condition of individual. Values for high income groups are close to those reported by Boyd. In pregnant females the increase in total serum lipids occurring in the first eight weeks of first trimester was more marked in the vegetarians than in the non-vegetarians. STC, ester cholesterol and free cholesterol showed the reverse trend. In the second trimester this difference was narrowed. In the third trimester there was no difference in the total serum lipid values between vegetarians and nonvegetarians, but there was now a slight increase for the nonvegetarians. Thus diet has no significant influence on lipid synthesis in the later period of pregnancy.

Green (1966) determined total cholesterol serially in a group of young women before and during pregnancy while

they consumed their usual diet or a fat modified diet known to have a hypocholesteroemic effect. During first trimester of pregnancy there was a slight but definite fall in serum cholesterol levels. After the first trimester, serum cholesterol levels increases gradually to peak at or near term. These changes occur both in normal and hypercholesterolemic females and is not affected by fat modified diets.

Hansen and co-workers studied 80 pregnant women and found no significant correlation between mothers intake of calories, proteins, fat and fatty acids to serum cholesterol, or fatty acid levels during third trimester.

Moses and his colleagues studied 65 young pregnant women from fifth month of pregnancy to term. 35 received ordinary institutional diet while 30 received same diet with 2 gm daily supplement of cholesterol. There was no significant difference in serum lipids of these two groups.

Arora snd Vinita (1987) studied the influence of dietary fat on serum total cholesterol level during antepartum intrapartum and postpartum periods of pregnancy and in the cord blood of neonate. They concluded that the levels of serum total cholesterol were higher in subjects, taking high fat diet, and lower in those taking normal and low fat diet. With advancement of pregnancy, during labour after delivery and in late postpartum period values were not statistically significant. However the cord blood serum total cholesterol values in relation to fat diet of mother

in third trimester were highly significant.

ROLE OF LACTATION DURING POSTPARTUM PERIOD

Hermann and Neumann (1918) studied that whole blood cholesterol and total lipids decreased during normal lactation but remained elevated when lactation did not occur. According to Boyd (1935) the lipid concentration of blood plasma was found to decline consistently after delivery in all cases where normal lactation occurred. The decline in values of plasma lipids during lactation was by and large due to loss of plasma neutral fat. After delivery, the elevated serum triglyceride concentration decreases rapidly and the significantly greater utilisation of serum triglyceride in lactating women could be caused by the tissue specific direction of VLDL towards the mammary gland for milk synthesis (Darmady et al, 1982). After neutral fats the greater decrease was found in phospholipids and next in free cholesterol and ester cholesterol. Changes in cholesterol fraction were comparatively alike.

Body reacts to normal lactation by having declining values for blood plasma lipids. Whether the blood plasma lipids fall during lactation because they are secreted in milk or because of the prior absence of certain hormones or other effects cannot be stated.

Few investigators have tried to correlate these effects with the activity of lipoprotein lipase enzyme. The enzyme activity is raised in adipose tissue during

early pregnancy in experimental animals and subsequently falls as gestation progresses. These changes are specific for the adipose tissue enzyme.

Evidence to support this hypothesis is provided by studies of postpartum rate (Hamosh et al, 1970; Scow et al, 1973). Lipoprotein lipase activity is depressed in adipose tissue but greatly elevated in mammary gland from suckling but not from non suckling rats.

The serum triglyceride concentration in suckling rats return to normal within few hours of delivery, but remains high for over 18 hours in non suckling rats. It is possible that the comparable pattern between lactating and nonlactating mothers in this study could have a similar explanation.

Several explanations have been offered as to where the excessive amounts of blood lipids move once pregnancy is over. It is proposed that in puerperium blood lipids are discharged through bile, urine and faeces (Hermann and Neumann) and in milk.

According to Boyd and most of the theories, fat content is a static value. Blood lipids are in equilibrium with tissue lipids. Lipids are removed from the blood, the loss is made up by the addition of lipids from the fat depots. It is unlikely that any removal except a very excessive one such as occurs in the increased metabolic rate of high fever (Boyd, 1935) could by itself account for lowering of values of lipids.

It is the factors which influence the equilibrium between blood and tissue lipids that we must seek to explain the effect of pregnancy and lactation. Boyd(1935) observed that in the puerperium this equilibrium is altered in the direction of a lowering of the level of plasma lipids, but of normal lactation is prevented, the change is inhibited or reversed.

PARTURITION - PHYSIOLOGY OF LABOUR

The act of child birth so simple to be thought is in itself a highly complex interaction of inumerable process and changes taking place with it. Parturition is the term used to define all these changes. It is infact a spectrum which can be divided into 3 phases, one emerging into another without a clear cut distinction.

1. Phase 1 : Preparation of uterus in the form of ripening of cervix and increased irritability of uterus.
2. Phase 2 : The onset of labour - including 3 stages of labour.

   Ist stage : Forceful uterine contractions established till full dilatation of cervix. Duration is 12 hours in primi and 6 hours in multigravida.

   2nd stage : From full dilatation of cervix to expulsion of foetus. Duration is 2 hours in primi and 30 minutes in multipara.

3rd stage : From expulsion of foetus to expulsion of placenta and membranes. Average duration 15 minutes in both primi and multipara.

3. Phase 3 : After delivery of placenta till complete involution of uterus.

ONSET OF PARTURITION

The various endocrine changes that occur during pregnancy and the various physiological and metabolic changes are interrupted with the onset of parturition. It is defined when late in pregnancy, there occurs an accelerated uterotropin-uterotonin formation and uterine changes that are preparatory to labour commence. Several hypothesis have been implicated for the onset of parturition and labour.

(i) PROGESTERONE WITHDRAWAL

Progesterone is responsible for the maintenance of quiescent uterus during pregnancy. Earlier it was believed that decrease in progesterone levels in maternal blood occurs before onset of parturition. However, it has been confirmed that no decrease in either the blood level or the production rate of progesterone occurs before or during labour. It occurs only with the delivery of placenta.

(ii) ALTERATION IN ESTROGEN AND PROGESTERONE RATIO

Though no drop in progesteron levels is noticed, but a definite alteration in the ratio between oestrogen

and progesterone occurs that is more important than absolute plasma concentration of these two hormones.

This alteration in oestrogen and progesterone ratio brings about the generation of an agent/agents that serve to effect increased hydrolysis of glycerophospholipids, release of free arachidonic acid and thence formation of prostaglandins. Also these changes alter the sensitivity of uterus to oxytocin and uterotounins at term.

(iii) ROLE OF CORTISOL

The pituitary adrenal axis of the fetus gives the signal for the initiation of labour.Increase in cortisol levels occurs which raises oestrogen levels. High cortisol levels have been observed in patients of spontaneous labour as compared to caeserian section. These are also increased in foetal and maternal stress during active labour. Cortisol might compete with progesterone receptors in myometrium by binding with the progesterone carrier protein.

An encephaly, postmaturity and adrenal hypoplasia is associated with prolonged gestation due to reduced cortisol levels due to reduced foetal adrenal function.

(iv) OXYTOCIN AND HUMAN PARTURITION

Oxytocin is one of the neurohypophysial hormone which is the most potent uterine stimulant at term. Increase in oxygocin level occurs during labour but there is no sudden spurt that would account for its role in

initiation of labour. The precise role is however, augmentive rather than initiative. It acts synergistically along with other hormones in achieving adequate uterine contractions necessary for expulsion of foetus.

(v) PROSTAGLANDINS

These are the ultimate uterine stimulant and probably the final link between complex neuroendocrine stimulus and their action at the target organs like uterus and cervix.

Prostaglandins are local hormones which are produced as a result of several stimuli like rupture of membranes, stretching of cervix and vagina. It acts on the contractile elements of myometrium, their release being stimulated by increase in ratio of oestrogen and progesterone and by uterine contractions.

During labour striking increase in concentration of both PGF_2 alpha and PGE_2 occurs in the amniotic fluid and maternal blood and urine.

LIPID METABOLISM IN FOETUS

Before birth, the fetus utilises carbohydrates as the major fuel for energy production. In utero transfer of glucose occurs through placenta. Thefetal glucose concentration follows the maternal closely although it is generally somewhat lower.

Free fatty acids have been shown to cross placenta but there is little or no transfer from mother to fetus of cholesterol, triglycerides or phospholipids. The synthesis of lipids in fetus proceed from glucose and fatty acid precursors in early stages of gestation and lipid content in fetus increases to 300 fold from first month to ninth month of gestation. After birth with cutting off of the nutrients from the maternal circulation and before milk feeding is started the newborn has to depend on its own endogenous sources of nutrients for survival.

Body lipids become a major source of energy for the newly born infant. Increased mobilisation of lipids from stores and increased lipolysis in the immediate postnatal period leads to a rise in the levels of total lipids, cholesterol, phospholipids and free fatty acids (Brown et al, 1939). The mechanism for the oxidation of fatty acids rapidly increases in activity after birth (Forfer, Ameil, 1984).

Hermann and Neumann (1912) for the first time measured the cord blood lipid levels and correlated it with the maternal levels. They observed that the cord blood cholesterol (62 mg/dl) values were considerably lower in comparison to their maternal counterpart (Mean value 264 mg/dl).

Sadowsky et al (1947) observed the cord and maternal cholesterol values in babies delivered normally.

Mean cord blood cholesterol values were 107 mg/dl which were higher than observed by earlier workers while the maternal mean blood cholesterol values were 262 mg/dl, which were comparable to earlier obtained values.

Rafstedt et al (1954) observed the cord blood cholesterol values to be 67 mg/dl.

Brown et al (1959) studied maternal serum during first stage of labour and cord blood samples collected just after birth. The mothers had normal full term delivery. The maternal values were 1104$\pm$172 mg%, 257$\pm$71 mg%, 847$\pm$176 mg% and 273$\pm$52 mg% for total lipids, lipoprotein lipids, betalipoprotein and cholesterol respectively, while they were 371$\pm$75 mg%, 147$\pm$40 mg%, 224$\pm$41 mg% and 82$\pm$17 mg% respectively for the same in the cord blood samples.

Brody and Carlson (1962) found that the concentration of serum triglycerides is quite low in the cord blood and newborn. This has been confirmed by Kaplan and Lee (1965) who had the same observations.

The authors have ascribed the differences in the maternal and cord blood lipid value to failure of the lipids to pass through the placental barrier. They also considered differences in fetal and adult metabolism to be responsible for the same, or perhaps the quiescent state of fat utilisation in the foetus and the absence of a need for fat mobilisation may also be responsible for low serum triglyceride concentration in the cord blood samples.

In cases of where the baby undergoes stress in utero or birth canal, increased triglyceride levels have been observed in the cord blood. This can be explained on the basis that early mobilisation and depletion of glycogen stores occurs and an early conversion to oxidation of fats results in increased triglyceride levels. Thus cord blood hypertriglyceridemia may be a useful indication of antepartum or intrapartum foetal stress or compromise.

The cord blood cholesterol or triglyceride values were associated with maternal fetal problem related with unfavourable intrauterine environment, fetal distress, fetal anoxia. There was a significant correlation between post term delivery and hypercholesterolemic neonates and low Apgar score along with maternal hypertension were more associated with hypertriglyceridemia. Low cord blood lipid levels were seen after an uneventful pregnancy, with the Apgar scores greater than 8. In babies of prolonged labour higher triglyceride levels were observed.

During birth newborn entered from a warm intra-uterine environment to unpleasant cool atmosphere and during this period of adjustment the energy requirements were provided by utilisation of carbohydrates and fat stores. The pituitary adrenocortical axis was supposed to be capable of stimulating fetal lipogenesis at term, and during stress of delivery catecholamines elicited an immediate response to adipose tissue. Neonatal stress

associated with maternofetal perinatal problems especially maternal hypertension and post term delivery. Low apgar scores were related to elevated cord blood cholesterol and triglyceride levels.

Cord blood cholesterol levels were not influenced by birth weight gestational age and elevated cholesterol level may indicate hypercholesterolemia. However, the levels of triglyceride and free fatty acids were affected by birth weight and gestation.

MATERIAL AND METHODS

MATERIAL AND METHODS

This study was carried out in the Department of Obstetrics and Gynaecology and the Lipid Research Laboratory of the Department of Medicine, M.L.B. Medical College, Hospital, Jhansi.

SELECTION OF CASES

Study comprised of pregnant females admitted to the antenatal wards or directly to the labour room. These patients were admitted for selective induction or termination or had already passed into labour and were in different stages of labour. 36 pregnant females were studied during the process of labour (PHASE I STUDY). With the ongoing work the study was extended to 21 females, after delivery but within 24 hours postpartum (PHASE II STUDY).

STUDY GROUP

The subjects were categorised into the following groups.

PHASE I STUDY (GROUP A)

Patients studied during intrapartum phase.

Total number of subjects studied were 36.

Mean age of patients was 24.9±4.1 years.

Mean weight of patients was 57±7 kg.

Gravid status - Primigravida : 22

- Multigravida : 14

Group 1 : Included normal pregnant females with spontaneous vaginal delivery (7 cases).

Group 2 : Normal pregnant females with vaginal delivery where artificial rupture of membranes was done to induce labour (6 cases).

Group 3 : Normal pregnant females with vaginal delivery where oxytocin augmentation was done(12 cases).

Group 4 : Normal pregnant females, who underwent elective caeserian section due to contracted pelvis, transverse lie or placenta praevia (7 cases).

Group 5 : Normal pregnant females who underwent Emergency Caeserian section due to non progress of labour or obstructed causes (4 cases).

PHASE II STUDY (GROUP B)

Patients studied during postpartum phase.

Total number of subjects studied - 21

Mean age of patients - 24.7±3.7 years.

Mean weight of patients - 56.4±5.2 kg.

Parity - Primipara : 11

Multipara : 10

Group 1B : Normal females who had delivered by vaginal route - spontaneous or induced (15 cases).

Group 2B : Normal females who underwent Emergency Caeserean section dur to prolonged labour, foetal distress, transverse lie(6 cases).

METHOD

History

A complete detailed history of the patients was taken with special emphasis on the following points -

- Age
- Socio-economic status.
- Present history : period of amenorrhoea.
  : Duration of onset of pains.
  : Any accompaniments e.g. leaking P/V
  : Any history of drug intake.
- Past history : It was ensured that the patient did not suffer from any disease which caused increased cholesterol levels such as coronary heart disease, kidney disease, liver disorder or diabetes mellitus and hypertension.
- Contraceptive history : Especially hormonal contraceptives.
- Menstrual history : LMP (Last menstrual period).
- Personal history : Diet with special reference to fat intake, addiction to tobacco etc.

EXAMINATION

General : Detailed examination with special emphasis upon :

- Built.
- Weight
- Blood pressure
- Pallor

Obstetrical

(i) Per Abdominal Examination (P/A) :

a. Fundal height - assessment and correlate it with the period of gestation. It was ascertained whether it corresponded to period of amenorrhoea.

b. Lie and presentation of the foetus in utero to certain cases this constituted an indication for selective caeserian section.

c. Amount of liquour assessed.

(ii) Per Vaginal Examination (P/V) :

- To know the presenting part.
- Assess the pelvis - detection of cases CPD.
- Progress and stage of labour.
- To time the blood samples according to the vaginal findings and to determine whether the patient will deliver vaginally or needs a caeserian section.

INVESTIGATIONS

Relevant investigations carried out :

Blood : Haemoglobin gm%.

: Blood group

Urine : Urine albumin

: Urine sugar

: Microscopic examination.

PERIOD OF COLLECTION OF BLOOD SAMPLES

PHASE I STUDY (GROUP A)

Blood samples were collected during the time when the process of labour had already started or was contemplated to start within a short duration. Following blood samples were taken.

1. In early labour - at the onset.
2. End of first stage - Full dilatation of cervix but before the delivery of baby.
3. End of second stage - After delivery of baby but before expulsion of placenta.
4. End of third stage - After delivery of placenta with the uterus fully contracted.

Corresponding to the blood samples, time and per vaginal findings were also noted so as to correlate them with each other. In patients of elective caeserean section sample No. 2 was not available as the patient does not pass into this stage. Similarly in patients of emergency section the patient was already near the end of stage I so sample No. 1 (above) could not be collected.

PHASE II STUDY (GROUP B)

Blood samples were collected in the postpartum phase.

First sample : At the end of stage III (0 hour). This corresponded to the sample No. 4(above).

Last sample : At 24 hours after delivery.

In between 2-3 samples were also taken from each patient at variable intervals from the end of third stage to within 24 hours postpartum. The samples were grouped as under :

1. 0 hours postpartum (P.P.)
2. 4 hours postpartum(P.P.)
3. 8 hours "
4. 12 hours "
5. 16 hours "
6. 20 hours "
7. 24 hours "

METHOD OF COLLECTION OF BLOOD SAMPLES

3-4 ml of blood was withdrawn from antecubital vein of the patient with minimal stasis in recumbent posture under all aseptic conditions. The sample was collected in dried autoclaved plain vials and allowed to settle for half an hour, so as to allow the blood to clot and sera to separate. It was then centrifuged. The serum collected was either subjected to investigations immediately or was preserved with standard precautions to

be investigated the next day. Thus attempt was made to study fresh sera only.

METHOD OF ESTIMATION OF VARIOUS LIPID PARAMETERS

The collrected samples were subjected to the following analysis.

1. Estimation of Serum Total Cholesterol (STC)

Serum total cholesterol estimation was done by Wynberg and Pileggi method (1970) utilising commercial kits supplied by Ethnor. Basic principle is that cholesterol reacts with solution of ferricperchlorate, ethyl acetate, and sulphuric acid to give a lavender coloured complex which can be measured calorimetrically at the optical density of 560-600 nm.

2. Estimation of serum Triglycerides (STG)

Serum triglyceride was estimated by acetyle acetone method. Glycerol that is released from fatty acids by saponification is oxidised to aldehyde by sodium meta-periodate. These aldehyde levels can be measured and are directly proportional to the amount of triglycerides.

3. Estimation of serum High Density Lipoproteins (HDL)

HDL was estimated utilising commercial kits supplied by Ethnor. Basic principle behind estimation is that HDL-c fraction is separated by precipitation using a precipitating agent. This precipitate when separated

contains chylomicrons, VLDL and LDL, which are removed by centrifugation. The supernatants contain HDL-c which is estimated by HDL-c colour reagent which gives a purple coloured complex which is measured calorimetrically at optical density of 560 nm. The intensity of colour is proportional to the concentration of HDL-c in the specimen under test.

4. Estimation of Serum Very Low Density Lipoproteins (VLDL)

VLDL is calculated by using the following formula given by Friedwald et al (1972). It is valid for STG values less than 400 mg%.

VLDL (mg%) = STG/5.

5. Estimation of Low Density Lipoproteins (LDL)

It is calculated by the following formula given by Fredrickson, DS (1972) :

LDL (mg%) = STC - (STG/5 + HDL)

= STC - (VLDL + HDL).

STATISTICAL METHOD USED

Student 't' test was used for the statistical analysis to compare the mean values in different groups.

O B S E R V A T I O N

The study was carried out in 2 phases. Total number of patients studied were 57. In phase I 36 patients were studied during the intrapartum phase, i.e. during the process of labour. In phase II study, carried in the post partum period, 21 patients were followed within 24 hours after delivery. These patients were not included in the phase I study. None of the patients were grossly obese. They were taking usual diet. Subjects of caeserean section were on IV fluids. None of the subjects studied had any medical disorder known to alter the lipoprotein profile.

PHASE I - GROUP A :

Comprised of patients studied during the intrapartum period. The patients were categorised under the following groups.

Group 1 : Patients who had normal spontaneous vaginal delivery without any intervention, served as control group.

Group 2 : Patients in whom ARM (Artificial rupture of membrane) was done to induce labour.

Group 3 : Patients, in whom oxytocin was given to augment labour.

Group 4 : Patients who underwent an elective caeserean section.

Group 5 : Consisted of patients who had an emergency caeserean section due to obstructed labour.

The age of patients was between 19 - 32 years with mean weight of 57$\pm$7 kg. Out of these 36 cases studied, 22 were primigravida and remaining 14 were multigravida.

TABLE I

Distribution of the number of patients studied during various stages of labour (Group A).

Group A	Early labour	End of the stage I	II	III
Group 1 (n=7)	7	6	6	6
Group 2 (n=6)	6	6	6	6
Group 3 (n=12)	12	12	8	12
Group 4 (n=7)	7	-	6	7
Group 5 (n=4)	-	4	4	4

The drop out in the number of subjects during labour was due to the sample having been haemolysed and hence erroneous results not considered.

Patient does not reach end of stage I in group 4.

Stage of early labour was absent in group 5 patients who presented at end of stage I.

TABLE II

Serum total cholesterol values during intrapartum phase in various groups (Mean ± S.D., mg/dl).

Group	A	Early labour	End of the stage I	II	III
Group	1	243.00 ±50.24	229.10 ±36.47	235.50 ±50.69	224.50 ±31.46
Group	2	209.17 ±39.47	196.30 ±37.00	221.69 ±23.85	211.00 ±22.85
Group	3	251.83 ±45.57	250.25 ±34.96	263.00 ±35.50	234.50 ±34.00
Group	4	246.80 ±37.90	-	226.60 ±34.45	221.70 ±40.65
Group	5	-	160.00 ±12.32	159.50 ± 8.43	185.50 ± 2.65

The above table shows a falling pattern between early labour and stage II in all the groups except in group 5 (Emergency LSCS) in which the levels increased steadily from the end of stage I to III and these values were statistically significant as shown below :

	't'	'p'
End of stage I Vs III	4.048	< 0.01
End of stage II Vs III	5.89	< 0.001

VARIATION IN STC VALUES BETWEEN THE GROUPS COMPARED DURING PROCESS OF LABOUR

Statistical analysis.

	Group 1 Vs 2		Group 1 Vs 3	
	't'	'p'	't'	'p'
Early labour	1.33	>0.05	0.39	>0.05
End of stage I	1.56	>0.05	1.189	>0.05
End of stage II	0.6125	>0.05	1.198	>0.05
End of stage III	0.861	>0.05	0.54	>0.05

	Group 1 Vs 4		Group 1 Vs 5	
	't'	'p'	't'	'p'
Early labour	0.161	>0.05	-	-
End of stage I	-	-	3.59	<0.01
End of stage II	0.3602	>0.05	2.91	<0.05
End of stage III	0.137	>0.05	3.04	<0.05

As evident from above analysis, no statistical significant difference was observed in STC values when group 1 (control group) was compared with 2, 3 and 4 at any stage of labour. The mode of termination does not affect the STC values during labour.

Group 5 had statistically significant low STC levels as compared to group 1.

TABLE III

HDL levels in different groups during intrapartum phase (Mean $\pm$.S.D, mg/dl).

Group A	Early labour	End of stage I	End of stage II	End of stage III
Group 1	47.57 ±10.08	47.00 ± 9.34	43.33 ± 8.36	42.67 ± 8.43
Group 2	43.16 ±12.04	41.83 ±12.23	46.83 ±14.75	46.50 ±14.37
Group 3	48.15 ±10.84	47.83 ±10.46	48.50 ±10.22	43.40 ± 9.53
Group 4	51.00 ±15.30	-	39.83 ± 6.49	42.42 ±12.81
Group 5	-	52.75 ± 5.74	48.25 ± 7.14	43.00 ± 8.64

There is no pattern of variation in HDL levels which remain relatively stable with no significant variations amongst various groups or during any stage of labour. This is given below:

Statistical analysis.

	Group 1 Vs 2 't'	Group 1 Vs 2 'p'	Group 1 Vs 3 't'	Group 1 Vs 3 'p'
Early labour	0.720	>0.05	0.237	>0.05
End of stage I	0.833	>0.05	0.164	>0.05
End of stage II	0.512	>0.05	1.008	>0.05
End of stage III	0.57	>0.05	0.1586	>0.05

	Group 1 Vs 4 't'	Group 1 Vs 4 'p'	Group 1 Vs 5 't'	Group 1 Vs 5 'p'
Early labour	0.499	>0.05	-	-
End of stage I	-	-	1.09	>0.05
End of stage II	0.820	>0.05	0.963	>0.05
End of stage III	0.040	>0.05	0.48	>0.05

No statistically significant alteration in HDL levels was noted between groups 1, 2, 3, 4 and 5.

TABLE IV

Serum triglycerides(STG) values during intrapartum phase in various study groups (Mean ± S.D., mg/dl).

Group A	Early labour	End of stage I	II	III
Group 1	125.40 ±35.13	113.00 ±27.53	113.50 ±20.33	108.00 ±16.31
Group 2	100.50 ±25.79	102.50 ±19.05	119.17 ±18.68	115.83 ±19.54
Group 3	126.08 ±37.88	122.58 ±33.96	141.31. ±25.88	113.16* ±30.76
Group 4	136.85 ±27.96	-	124.00 ±29.60	118.85 ±18.96
Group 5	-	92.50 ± 3.79	99.00** ± 4.16	105.30 ±11.00

* Statistically significant as compared to stage II ($p < 0.05$) of same group.

** Statistically significant as compared to group 3 in the same stage ($p < 0.01$).

The STG levels fell between early labour and stage II but this difference is insignificant except in group 3 where oxytocin had been used to augment labour a significant fall occurs between end of stage II and III.

Statistical analysis of Table IV.

	Group 1 Vs 2 't'	Group 1 Vs 2 'p'	Group 1 Vs 3 't'	Group 1 Vs 3 'p'
Early labour	1.434	>0.05	0.039	>0.05
End of stage I	0.766	>0.05	0.597	>0.05
End of stage II	0.2666	>0.05	2.161	>0.05
End of stage III	0.763	>0.05	0.381	>0.05

	Group 1 Vs 4 't'	Group 1 Vs 4 'p'	Group 1 Vs 5 't'	Group 1 Vs 5 'p'
Early labour	0.680	>0.05	-	-
End of stage I	-	-	1.45	>0.05
End of stage II	0.72	>0.05	1.38	>0.05
End of stage III	1.096	>0.05	0.287	>0.05

No alteration in STG values was noted amongst various groups. Thus again mode of delivery does not produce any significant change in STG levels during labour.

TABLE V

Serum low density lipoprotein (LDL) values during intrapartum phase in various groups (Mean ± S.D., mg/dl).

Group A	Early labour	End of stage I	End of stage II	End of stage III
Group 1	170.34 ±38.40	159.56 ±27.03	169.46 ±48.05	160.23 ±30.00
Group 2	145.90 ±34.83	134.00 ±32.69	151.00 ±24.14	141.33 ±20.31
Group 3	177.87 ±34.34	169.40 ±33.89	185.07 ±24.35	168.53 ±31.46
Group 4	168.49 ±31.34	-	162.03 ±33.70	155.51 ±33.68
Group 5	-	88.75 ± 6.40	91.45 ± 2.64	121.50 ± 8.25

LDL values similar to STC fall between early labour to stage III. None of these values were found to be statistically significant at any stage of labour in all groups except group 5 as observed from table V.

In patients of obstructed labour undergoing emergency LSCS, there was an increase instead of the fall as noted in other groups and this change was statistically significant as depicted below :-

<u>Group 5</u>

Stage I Vs II ($t = 6.468$, $p < 0.001$)

Stage I Vs III ($t = 6.216$, $p < 0.001$).

Statistical analysis of Table V.

	Group 1 Vs 2		Group 1 Vs 3	
	't'	'p'	't'	'p'
Early labour	1.193	>0.05	0.44	>0.05
End of stage I	1.494	>0.05	0.616	>0.05
End of stage II	0.852	>0.05	0.799	>0.05
End of stage III	1.294	>0.05	0.535	>0.05

	Group 1 Vs 4		Group 1 Vs 5	
	't'	'p'	't'	'p'
Early labour	0.099	>0.05	-	-
End of stage I	-	-	5.05	<0.001
End of stage II	0.314	>0.05	3.18	<0.05
End of stage III	0.294	>0.05	2.477	<0.05

Patients of obstructed labour (group 5) showed significant low values of LDL as compared to group 1 in all the stages studied. No difference can be seen in any of the other groups.

PHASE II STUDY (GROUP B)

Patients were studied during the postpartum phase of pregnancy. A total of 21 patients were included with mean age of 24.7±3.7 years and mean weight of 56.4 ±5.2 kg. Out of total 21 patients, 15 cases delivered vaginally both spontaneous and induced deliveries included (group 1B) and 6 cases delivered by emergency caeserean section due to foetal distress, prolonged labour or transeverse lie in advanced labour (Group 2B). Eleven cases were primipara and remaining 10 cases were multipara.

TABLE VI

Distribution of the number of patients studied during the postpartum period in hours after end of stage III.

Groups	Total No.of cases	Postpartum periods (hours)						
		0	4	8	12	16	20	24
1B	15	15	6	6	7	6	6	15
2B	6	6	3	3	3	3	3	6

Blood samples were taken at 0 and 24 hour post-partum from each patient. 2-3 more samples were also taken from same patient at varying periods and later grouped according to the duration in hours post partum.

TABLE VII

Values of lipid parameters(STC, HDL, STG and LDL) during the post partum phase in patients of group 1B (Mean ± S.D., mg/dl).

Postpartum period(hours)	STC	HDL	STG	LDL
0 (End of stage III)	233.50 ±17.50	48.20 ± 6.82	116.80 ±13.54	162.06 ±10.58
4 hours P.P.	232.60 ±17.23	47.60 ± 5.55	108.40 ±12.35	163.32 ±13.21
8 hours P.P.	227.60 ±14.50	46.30 ± 6.43	106.80 ±11.62	159.94 ±10.75
12 hours P.P.	210.50 ±16.95	42.10 ± 5.19	101.60 ±10.23	148.08 ±14.42
16 hours P.P.	208.30 ±20.40	40.50 ± 6.20	98.10 ±16.18	148.18 ±10.96
20 hours P.P.	206.20 ±20.60	39.60 ± 4.14	96.20 ±13.05	147.60 ±10.55
24 hours P.P.	204.60 ±20.10	39.40 ± 4.51	93.40 ± 8.64	146.60 ±13.92

TABLE VIII

STC values in group 1B during postpartum phase (Mean ± S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean ±S.D.	233.5 ±17.5	232.6 ±17.23	227.6 ±14.5	210.5 ±16.95	208.3 ±20.4	206.2 ±20.6	204.6 ±20.1

Table XVIII shows that STC declined in the post partum phase from 0-24 hours. There was a fall of 12.5% in STC levels within 24 hours.

Statistical analysis of table VIII.

	't'	'p'
0 - 4 hours PP	0.059	>0.05
0 - 8 hours PP	0.728	>0.05
0 - 12 hours PP	2.74	<0.05
0- 16 hours PP	2.85	<0.01
0 - 20 hours PP	3.078	<0.01
0 - 24 hours PP	4.202	<0.001

It clearly shows the significant fall in STC levels at 12 hours postpartum. The falling levels becoming increasingly significant ($p < 0.05$ at 0-12 hours) to highly significant ($p < 0.001$ at 0-24 hours).

TABLE IX

HDL values in group 1B during postpartum period (Mean $\pm$ S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean $\pm$S.D.	48.2 $\pm$6.82	47.6 $\pm$5.55	46.3 $\pm$6.43	42.1 $\pm$5.19	40.5 $\pm$6.20	39.6 $\pm$4.44	39.4 $\pm$4.51

HDL values fall during the postpartum phase as shown in table IX.

Statistical analysis of table IX.

	't'	'p'
0 - 4 hours PP	0.216	>0.05
0 - 8 hours PP	0.610	>0.05
0 - 12 hours PP	2.095	<0.05
0 - 16 hours PP	2.393	<0.05
0 - 20 hours PP	2.862	<0.01
0 - 24 hours PP	4.171	<0.001

The lowered HDL values become significant at 12 hours postpartum and highly significant at 24 hours.

TABLE X

STG values in group 1B during postpartum phase (Mean ± S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean ±S.D.	116.8 ±13.54	108.4 ±12.35	106.8 ±11.62	101.6 ±10.23	98.1 ±16.18	96.21 ±13.05	93.4 ± 8.64

The above table shows the declining pattern of values of STG in puerperium. A fall of 20.03% occurs in STG levels within 24 hours postpartum.

Statistical analysis of table X.

	't'	'p'
0 - 4 hours PP	1.314	>0.05
0 - 8 hours PP	1.58	>0.05
0 - 12 hours PP	2.63	<0.05
0 - 16 hours PP	2.71	<0.05
0 - 20 hours PP	3.18	<0.01
0 - 24 hours PP	5.648	<0.001

The falling STG levels in the postpartum phase became significantly low at 12 hours and thereafter the fall continues.

TABLE XI

LDL values in group 1B during postpartum phase (Mean ±S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean ±S.D.	162.06 ±10.58	163.32 ±13.21	159.04 ±10.75	148.08 ±14.42	148.18 ±10.96	147.60 ±10.55	146.60 ±13.92

As with other parameters LDL values also have a decreasing trend in the postpartum phase (Table XI).

Statistical analysis of table XI.

	't'	'p'
0 - 4 hours PP	0.2302	>0.05
0 - 8 hours PP	0.4180	>0.05
0 - 12 hours PP	2.440	<0.05
0 - 16 hours PP	2.690	<0.05
0 - 20 hours PP	2.819	<0.05
0 - 24 hours PP	3.413	<0.01

The fall in LDL levels became significant at 12 hours.

TABLE XII

Values of lipid parameters (STC, HDL, STG and LDL) during post partum phase in patients of caeserean section (Group 2B) (Mean$\pm$S.D., mg/dl).

Postpartum period (hours)	STC	HDL	STG	LDL
0 (End of stage III)	195.50 $\pm$ 7.10	47.10 $\pm$ 3.20	112.60 $\pm$ 4.60	126.25 $\pm$ 3.20
4 hours P.P.	186.30 $\pm$ 6.90	43.27 $\pm$ 3.24	108.70 $\pm$ 5.40	121.29 $\pm$ 4.60
8 hours P.P.	182.30 $\pm$ 6.83	41.02 $\pm$ 4.58	102.30 $\pm$ 4.49	120.82 $\pm$ 3.37
12 hours P.P.	177.40 $\pm$ 5.60	39.16 $\pm$ 6.58	100.50 $\pm$ 5.20	118.14 $\pm$ 5.20
16 hours P.P.	175.00 $\pm$ 6.42	37.42 $\pm$ 5.31	99.20 $\pm$ 5.73	117.74 $\pm$ 5.60
20 hours P.P.	172.60 $\pm$ 6.00	35.90 $\pm$ 6.32	97.90 $\pm$ 4.03	117.12 $\pm$ 4.30
24 hours P.P.	170.60 $\pm$ 5.40	36.30 $\pm$ 4.88	96.40 $\pm$ 5.80	115.82 $\pm$ 4.00

TABLE XIII

STC levels in group 2B during postpartum period (Mean ± S.D., mg/dl).

	Postpartum period(hours)						
	0	4	8	12	16	20	24
Mean ±S.D.	195.50 ± 7.10	186.30 ± 6.90	182.30 ± 6.83	177.40 ± 5.60	175.00 ± 6.42	172.60 ± 6.00	170.60 ± 5.40

STC levels fell in the puerperium. A fall of 12.7% was noted within 24 hours in the postpartum period.

Statistical analysis of table XIII.

	't'	'p'
0 - 4 hours P.P.	1.8483	-
0 - 8 hours P.P.	2.6596	$\angle 0.05$
0 - 12 hours P.P.	3.821	$\angle 0.01$
0 - 16 hours P.P.	4.196	$\angle 0.01$
0 - 20 hours P.P.	4.763	$\angle 0.01$
0 - 24 hours P.P.	6.8498	$\angle 0.001$

The falling values became statistically significant at 0-8 hours postpartum and the fall continued till 24 hours.

TABLE XIV

HDL levels in group 2B during postpartum period (Mean $\pm$ S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean $\pm$S.D.	47.10 $\pm$3.20	43.27 $\pm$3.24	41.02 $\pm$4.58	39.16 $\pm$6.58	37.42 $\pm$5.31	35.90 $\pm$6.32	36.30 $\pm$4.88

A pattern of fall was observed in HDL levels throughout 24 hours postpartum.

Statistical analysis of table XIV.

	't'	'p'
0 - 4 hours PP	1.701	>0.05
0 - 8 hours PP	2.367	<0.05
0 - 12 hours PP	2.54	<0.05
0 - 16 hours PP	3.503	<0.01
0 - 20 hours PP	3.668	<0.01
0 - 24 hours PP	4.976	<0.001

As clear from the above the HDL values became statistically low at 8 hours postpartum.

TABLE XV

STG levels in group 2B during post-partum period (Mean $\pm$ S.D., mg/dl).

	Post partum period (hours)						
	0	4	8	12	16	20	24
Mean $\pm$S.D.	112.60 $\pm$ 4.60	108.70 $\pm$ 5.40	102.30 $\pm$ 4.49	100.50 $\pm$ 5.20	99.20 $\pm$ 5.73	97.90 $\pm$ 4.03	96.40 $\pm$ 5.80

STG levels decreased in the postpartum phase as is clear from the above table.

Statistical analysis of table XV.

	't'	'p'
0 - 4 hours P.P.	0.555	>0.05
0 - 8 hours P.P.	2.568	<0.05
0 - 12 hours P.P.	2.988	<0.05
0 - 16 hours P.P.	3.264	<0.05
0 - 20 hours P.P.	4.045	<0.01
0 - 24 hours P.P.	4.374	<0.01

The values of STG became significantly low at 8 hours and the fall continued till 24 hours postpartum.

TABLE XVI

LDL levels in group 2B during post-partum period (Mean ± S.D., mg/dl).

	Postpartum period (hours)						
	0	4	8	12	16	20	24
Mean ±S.D.	126.25 ± 3.20	121.29 ± 4.60	120.82 ± 3.37	118.14 ± 5.20	117.74 ± 5.60	117.12 ± 4.30	115.02 ± 4.00

As with the other parameters the LDL levels also fell in the postpartum phase. The values became significantly low at 8 hours postpartum as is shown in statistical analysis given below.

Statistical analysis of table XVI.

	't'	'p'
0 - 4 hours P.P.	1.9259	>0.05
0 - 8 hours P.P.	2.371	<0.05
0 - 12 hours P.P.	2.960	<0.05
0 - 16 hours P.P.	2.990	<0.05
0 - 20 hours P.P.	3.647	<0.01
0 - 24 hours P.P.	4.501	<0.01

D I S C U S S I O N

D I S C U S S I O N

The present study is concentrated to the changes in the lipoprotein profile during the process of labour and thereafter within twenty four hours postpartum. Normal healthy pregnant females who delivered spontaneously served as a control group. Lipoprotein profile was studied in patients who delivered after induction (ARM oxytocin) and also in patients of elective and emergency caeserean sections.

Observed basal lipoprotein profile values at the onset of labour was STC 243 ± 50.24 mg%, HDL-c 47.57 ± 10.08 mg%, STG 125.4 ± 35.13 mg%, LDL 170.34 ± 38.40 mg% in patients who delivered spontaneously (control group - group 1). No significant difference was observed amongst various groups demonstrating the homogenecity of the selected cases. In group 5 when the patient came at the end of stage I comparable data for early labour values was not available.

SERUM TOTAL CHOLESTEROL (GROUP A)

The levels ranged between 209-251 mg% in the various groups during early labour.

The values tend to fall by the end of stage III during labour in all the groups but this fall is insignificant. Group 5 patients who are in obstructed labour for more than 24 hours presented a different picture.

STC levels were significantly lower than any of the other groups at the end of stage I and the values unlike the other groups showed an increase from the time of admission (end of stage I) to the end of stage III to an extent of 15.9% ($p \angle 0.01$). The different presentation of patients in obstructed labour cannot be accounted for the effect of stress. Had it been so patients of elective caeserean section where process of labour is curtailed would have presented differently. The possible explanation can be that the regulatory mechanisms in the body ensure a supply of suitable fuel for all tissues at all times, from the fully fed to the totally starved state. Breakdown of these mechanisms occurs owing to hormonal imbalance during pregnancy. Hence starvation or fasting state as observed in patients of obstructed labour (more than 24 hours) results in lowered glucose availability, reduced insulin levels, increased glucagon levels, reduced HMG Co A activity and hence reduced cholesterol synthesis. Moreover, reduced insulin levels cause reduction in the inhibition of lipogenesis, hence increased lipolysis and increased free fatty acid levels. Insulin in an absolute or relative deficient state also abolishes the usual antilipolytic activity of PGE_1 (Circulating levels of PGE_1 are raised during labour) and increased lypolysis. In these patients the contracting uterus continues to utilize glucose, the fate of FFA is altered which instead of esterification are oxidised and

subsequently low STG levels results. The recovery of the STC levels (indicated by an increase through the different stages of labour) results due to the intravenous fluids (10%, 25% glucose) rich in glucose, which on one hand alters the metabolism of FFA to esterification instead of oxidation resulting in increased STG levels. At the same time insulin release is stimulated to cause inhibition of lipolysis and return of antilipolytic activity of PGE_1 and increased STC levels.

Another possible explanation for the low STC levels in patients of obstructed labour can be that they were all primigravidae. Potter et al (1979) have noted significant ($p < 0.05$) low STC levels in primigravidas as compared with multigravidas.

SERUM TOTAL CHOLESTEROL (GROUP B)

In the postpartum phase the elevated STC levels fell significantly ($p < 0.05$) at eight hours postpartum in patients of caeserean section and at 12 hours in patients of normal vaginal delivery.

The possible explanation to this delayed fall in patients who had a vaginal delivery could be the early resumption of oral intake of food. Patients of caeserean section were kept nill orally and maintained on intravenous fluids. It is known that the postpartum hyperlipidemia is found to be very sensitive to dietary manipulation, responding quickly to changes in cholesterol content and

fatty acid composition (Potter & Nestel, 1976).

The fall in STC continues and the values became highly significant ($p < 0.001$) at 24 hours postpartum in both the groups. A 12.5% fall was noted in either groups. Potter and Nestel (1979) have also noted a fall of 14% within 12 to 24 hours of delivery in STC levels.

Since the rise in lipid levels in pregnancy have been attributed to the placental hormones, it may be worthwhile looking at the half life profile of these hormones. HPL, oestriol and conjugated oestrogens are elaborated by the placenta. According to Klopper (1978) by eight hours postpartum the total oestriol levels (the main circulating hormone during pregnancy) falls to 33%, unconjugated oestriol fall to 20% whereas conjugated oestrogens fall to 33% of its basal intrapartum values. HPL falls steeply and by 3 hours virtually none can be found in circulation. Therefore we presume that our fall in lipids is related to the sharp decline in placental hormones.

<u>LOW DENSITY LIPOPROTEINS (LDL) : (GROUP A)</u>

The LDL levels also followed the same variations as the STC levels in patients of group 5 with a significant ($p < 0.001$) increase from stage I to stage III. In other groups no such alteration was observed though the exact values tend to decrease from 170.34 ± 38.40 mg% at onset of labour to 160.33 ± 30.00 at the end of stage III with no significant intergroup variations.

GROUP B

The LDL levels demonstrated similar changes as the STC levels in the postpartum phase. The values decreased from 162.06±10.58 to 146.60±13.92 mg% within 24 hours in patients of vaginal delivery ($p < 0.01$). In patients of caeserean section also the significant fall ($p < 0.01$) occurs within 24 hours postpartum, fall becoming significant ($p < 0.05$) earlier in the latter group.

HIGH DENSITY LIPOPROTEIN (HDL)

GROUP A

HDL fraction did not alter during the process of labour in any of the groups studied. The observed mean values ranged from 51.00±15.30 to 43.16±12.04 mg% during the stage of early labour. There being no intergroup or interstage significant variations.

GROUP B

During the postpartum phase the values of HDL fell significantly ($p < 0.001$) to the extent of 18.2% in patients of vaginal delivery at the end of 24 hours. The falling levels becoming significant at 12 hours ($p < 0.05$) to highly significant at 24 hours ($p < 0.001$). Similar to that observed for STC levels. The change occurring earlier in patients of caeserean section. As discussed earlier these changes are possibly related to the falling hormonal levels.

SERUM TRIGLYCERIDES (STG)

STG levels showed an increase in the values between early labour and stage II, followed subsequently by a fall in stage III. This fall was significant in patients of group 3 in whom oxytocin had been used to augment labour. No referral data for STG levels during labour is available. Fairweather et al (1965) studied the NEFA levels and not the STG levels. They have concluded that the use of oxytocin to induce labour does not alter the normal pattern of NEFA when given in a physiological range but the simultaneous administration of glucose, duration of labour, duration and dosage of oxytocin would alter the levels. The NEFA levels do not directly reflect the STG levels. The relation between NEFA and STG needs to be studied.

In our study the STG levels in the control group 1 were 125.40±35.13 mg% during early labour, 113.00±27.53 mg% at the end of stage I, 113.50±20.33 mg% and 108.0± 16.31 mg% at the end of stage II and III respectively. None of the differences in values were statistically significant in any other group.

Patients of emergency caeserean section (group 5) exhibited least STG levels when compared to the other groups though this difference was not statistically significant. Previous study made by Neeta et al (1993) have demonstrated higher STG levels during intrapartum

phase in patients of emergency LSCS and have attributed these high levels to perinatal stress and prolonged labour. In our study the values obtained were low.

In group 5 patients there was a rise in the STG levels in contrast to the other groups where a fall has been noted, although this increase is not statistically significant - from 92.50$\pm$3.79 mg% at the end of stage I to 105.30$\pm$11.00 mg% at the end of stage III.

Potnis et al (1977) have attributed that changes occur during labour as a result of stress which in turn causes increase in catecholamines which alter the oestrogen: progesterone ratio and the release of prostaglandins. Labour is a stressful condition but in our study we did not encounter any significant alteration in any of the lipid parameters through the various stages of labour.

<u>SERUM TRIGLYCERIDES (GROUP B)</u>

In the postpartum phase STG levels fell in both the groups, the maximum fall being of 20.03% (from 116.80 $\pm$13.54 to 93.40$\pm$8.64) in patients of vaginal delivery at 24 hours.

Watson (1957), De Alvarez et al (1959), Konttinen et al (1964) have all shown a fall in triglyceride levels in puerperium. Potter and Nestel (1979) have shown a fall of 24% in plasma triglycerides in 24 hours and also triglycerides fall more steeply than

cholesterol. In our study the fall was 12% for cholesterol whereas Potter and Nestel (1979) reported a fall of 14%.

We found that the fall in triglycerides is statistically significant at 12 hours puerperium. We attribute this fall to the lack of placental hormones (see earlier discussion on cholesterol). Dannenberg et al (1962) have shown that STG levels increase during the first 24 hours however they have not provided any explanation. In patients of LSCS this fall occurs earlier but is slow as compared to patients delivered by vaginal route.

To conclude plasma lipids increase during pregnancy reaching a peak at the end of third trimester. They remain at this high level during the process of labour with minor variations and then rapidly fall between 12 to 24 hours to achieve statistical significance. The fall then continues for the rest 6-7 weeks.

SUMMARY AND CONCLUSION

SUMMARY AND CONCLUSION

1. Lipoprotein profile changes were studied during the intrapartum and postpartum phases of pregnancy.
2. 36 patients in the age group between 19-32 years were studied during the intrapartum phase (Group A). They were categorised under the following groups.
   1. Spontaneous delivery, 2 in whom ARM had been done to induce labour, 3. Oxytocin used to induce labour, 4. Patients who underwent elective caeserean section, 5. Patients of emergency caeserean section due to obstructed labour.
3. Lipoprotein profile (STC, HDL, STG, LDL) was studied in all the groups at early labour and end of stage I, II. and III.
4. 21 patients during the postpartum phase (Group B) were studied under two groups. 1. patients who had normal vaginal delivery, 2. Patients who had caeserean section. Patients of group A were not included in this group.
5. Lipoprotein profile was studied at the end of stage III (0 hours) and then at 4 hourly intervals till twenty four hours puerperium in group B.

The following conclusions were made :-

CHANGES IN LIPOPROTEIN PROFILE - INTRAPARTUM (GROUP A)

a. The process of labour does not bring about any significant change in the lipoprotein profile.

b. STC, HDL, STG and LDL do not undergo any significant alteration during the intrapartum phase in patients of vaginal delivery both spontaneous and induced and in patients of elective LSCS. However, in patients of emergency caeserean section due to obstructed labour. STC and LDL values are low at end of stage I and the values increase significantly during the intrapartum phase. In patients in whom oxytocin had been used to augment labour. STG showed a significant fall between end of stage II and end of stage III.

c. Mode of delivery does not significantly alter the lipid profile during the intrapartum period.

CHANGES IN LIPOPROTEIN PROFILE POSTPARTUM (GROUP B)

a. Fall in all the lipid parameters occurs after end of stage III and gains significance at 8 hours in patients of caeserean section and at 12 hours in patients of vaginal delivery.

b. At 24 hours the fall in lipoprotein fractions is highly significant in both groups.

B I B L I O G R A P H Y

B I B L I O G R A P H Y

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